Pneumatosis Intestinalis in Patients With Severe Thermal Injury

Todd F.M. Huzar, MD, John Oh, MD, Evan M. Renz, MD, Steven E. Wolf, MD, Booker T. King, MD, Kevin K. Chung, MD, Christopher E. White, MD, Edward Malin, MD, Jonathan B. Lundy, MD, Seung H. Kim, MD, Lorne H. Blackbourne, MD, Leopoldo C. Cancio, MD

Severe thermal injury is associated with pronounced changes in intestinal physiology, which may cause ischemia, infarction, and pneumatosis intestinalis (PI). PI is a pathologic condition defined as infiltration of gas into the gastrointestinal tract wall. Historically, PI prompted urgent surgery, yet some surgeons "watch and wait" to avoid the risks of a negative laparotomy. The authors reviewed experience with PI at a single burn center. They retrospectively identified burn center intensive care unit patients with radiographic or pathologic evidence of PI. Data included demographics, injury severity score, TBSA burned, operative findings, length of stay, and mortality. From January 2003 through August 2009, 1129 patients were admitted to the authors' burn center intensive care unit. Fifteen had PI. Twelve had radiographic evidence of PI, and 10 had PI associated with intestinal infarction. Nonsurvivors had lower base deficits ($P = .02$), higher lactate levels ($P = .05$), and required vasopressor support ($P = .02$) within 24 hours of developing PI. Massive intestinal infarction ($P = .004$) and open abdomens ($P = .004$) were more common among nonsurvivors. PI can be identified by radiologic or pathologic findings. The authors' experience with PI among patients with burn injury revealed a high mortality rate. Because of the association of bowel ischemia with PI, exploratory laparotomy should be strongly considered in patients with burn injury with radiographic evidence of PI. (J Burn Care Res 2011; 32:e37–e44)

Pneumatosis intestinalis (PI) is defined as the presence of gas or gas-filled cysts within the bowel wall, a phenomenon first described by DuVernoi[1] in autopsy studies in 1783. A more comprehensive description of PI was published by Bang[2] in 1876. It was not until the late 1940s that Gazin et al[3] reported the first case of PI diagnosed preoperatively using radiographs and confirmed by laparotomy and through microscopic examination of the specimen. In 1952, Koss[4] categorized the condition into primary (15%) and secondary (85%) causes. Primary PI was signified by gas-filled cysts, whereas secondary PI was attributed to underlying gastrointestinal (GI) disease.[1,5]

The true incidence of PI is unknown, because patients may remain asymptomatic. It is usually identified in symptomatic patients through radiologic evaluation, but diagnosis may be made at the time of operation or from a pathology specimen. Even in the presence of symptoms, the clinical significance of PI is variable, making its relationship with GI disease and its management controversial.

Like many other clinical challenges, management of PI in adults is often guided by a surgeon's experience. Typically, radiologic findings of PI are associated with bowel infarction, a need for emergent operative intervention, and a high mortality rate. However, some surgeons refrain from a policy of urgent operation for all patients and endorse the concept of "watch and wait" to avoid unnecessary lapa-

From the United States Army Institute of Surgical Research, Fort Sam Houston, Texas.

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Address correspondence to Leopoldo C. Cancio, MD, United States Army Institute of Surgical Research, 3400 Rawley E Chambers Avenue, Building 3611, Fort Sam Houston, Texas 78234-3611.

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rotomy, invoking the success of such a strategy in infants with necrotizing enterocolitis.[6] Currently, no compelling evidence exists to definitively answer the question of whether to operate in the face of PI seen on radiographs.

Patients sustaining severe burns are at risk for many complications including those related to the GI system. Marked fluid shifts, dramatic changes in cardiac output, and decreased regional organ perfusion may contribute to intestinal ischemia and infarction. Desai et al[7] and Kowal-Vern et al[8] reported the effects of severe thermal injury on the GI tract and the increased risk of abdominal catastrophes such as mesenteric ischemia and bowel infarction. It was further established that abdominal catastrophes may develop in patients with burn injury who have not sustained abdominal trauma.[9] PI can occur in any critically ill patient population, and patients with burn injury are no exception. To date, the incidence of PI in patients with burn injury is unknown, and only a few cases have been reported.[8,10]

The purpose of this study was to examine the incidence of PI in patients admitted to our burn center and to determine whether radiographic evidence of PI indicates the need for operative intervention for ischemic bowel in these patients. We sought to review our management of patients with PI, the relationship between PI and bowel ischemia, and its relationship with mortality.

METHODS

We performed an institutional review board–approved review of patients admitted to the US Army Institute of Surgical Research burn center intensive care unit (BICU) from January 1, 2003, to August 31, 2009. We restricted our search to adult patients (18 years or older) with PI identified by radiologic, intraoperative, or histopathologic methods in our burn registry, electronic medical records, and pathology reports. We reviewed demographic data (age, gender, and military status), admission and injury-specific data (%TBSA, % full-thickness burned, presence of inhalation injury, injury severity score, date of injury, and date of admission), radiologic findings (type of study, date of diagnosis, location of PI, and presence of hepatic portal venous gas [HPVG]), clinical data before and after diagnosis (lactate and base deficit levels, vasopressor use, and enteral feeding rate), intraoperative data (findings, extent and location of PI and ischemia, correlation with preoperative imaging, and operative interventions), pathology results, and outcomes.

Burn size was described as TBSA in the patient's medical record at the time of admission. Laboratory data represented the most severe value within the 24 hours preceding the diagnosis. Vasopressor use was defined as the infusion of a vasoactive agent within 24 hours of the diagnosis of PI or ischemic bowel. Radiologic diagnosis of PI was defined as the presence of intramural gas or HPVG on plain films or CT scans (Figures 1 and 2). HPVG was included in this study as a "positive" finding because of its known association with PI and intestinal ischemia.[11] The time of clinical diagnosis of an abdominal catastrophe was determined from surgeon's preoperative notes or from radiology reports in the electronic medical record. A laparotomy was considered positive if there was gross evidence of intestinal ischemia or if pathologic review of specimens demonstrated PI. The extent of infarc-

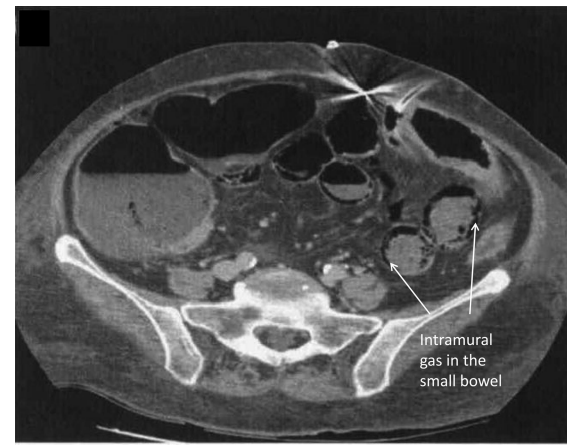

Figure 1. Pneumatosis intestinalis of the small bowel demonstrated on CT scan of the abdomen by intramural gas (arrows).

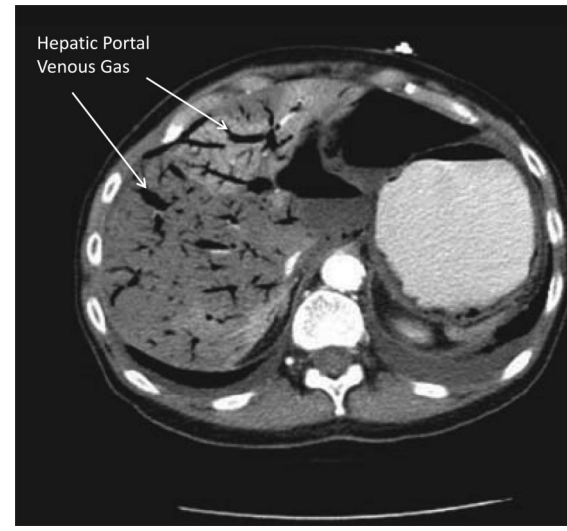

Figure 2. CT scan demonstrating hepatic portal venous gas (arrows) in a patient with pneumatosis intestinalis.

tion identified at laparotomy was categorized into segmental or massive. Segmental infarction was defined as infarction limited to 30 cm or less in the small bowel or limited to a single segment of large bowel. Massive infarction was defined as infarction of more than 30 cm of small bowel or of two or more large bowel segments. Enteral feeding within 48 hours before diagnosis of PI was noted. During this study period, a variety of enteral tube feeding formulas were used with different caloric contents, ranging from 1.2 to 2.0 kcal/ml. Dyes were not used to color the formulas.

Statistical analysis was performed with Statistical Analysis Software (SAS Institute Inc., Cary, NC) and Microsoft Excel 2007 (Microsoft Inc., Redmond, WA). Categorical data were analyzed using Fisher's exact test. Continuous data were analyzed using Wilcoxon two-sample, Mann-Whitney, and Kruskal-Wallis tests. Statistical significance was defined as $P < .05$.

RESULTS

Of the 1129 patients admitted to the BICU during the study period, 337 were military casualties and 792 were civilians. Fifteen (1.3%) developed PI during their admission. The majority of patients developing PI were men (n = 13, $P = .47$). Demographic data for survivors and nonsurvivors are summarized in Table 1. Patients with PI had a mortality rate of 73%. There was no statistical difference between survivors and nonsurvivors with respect to age, TBSA, full-thickness burned, and inhalation injury.

Table 1. Demographic data

	Nonsurvivors (n = 11)	Survivors (n = 4)	Overall (n = 15)	*P*
Age (yr)	28 (22–45)	21 (20–25)	27 (21–44)	.09
Male gender (%)	10 (91)	3 (75)	13 (87)	.47
TBSA	52 (42–72)	27 (22–37)	51 (33–58)	.08
FT	47 (17–63)	14 (8–26)	41 (13–50)	.19
ISS	26 (25–34)	34 (33–38)	29 (25–34)	.12
Inhalation injury (%)	9 (81)	2 (50)	11 (73)	.52
CRRT (%)	5 (45)	1 (25)	6 (40)	1.00
Date of injury to PI (d)	8 (7–40)	15 (9–24)	10 (8–28)	.89
Date of admit to PI (d)	7 (5–39)	11 (9–17)	8 (6–26)	.74

Values are medians (25th and 75th interquartile range).
ISS, injury severity score; *CRRT*, continuous renal replacement therapy; *FT*, % full-thickness area burned; *PI*, pneumatosis intestinalis.

In general, survivors demonstrated PI on preoperative radiographic studies and underwent laparotomy as a result of these findings. Thirteen patients (87%) underwent radiologic evaluation in the perioperative period as a result of a tube feed intolerance (10/15), worsening abdominal distension (15/15), severe metabolic acidosis, and concerns for intestinal ischemia. Studies in 12 patients were deemed positive and had the following radiographic findings: PI only (6 patients), HPVG only (2 patients), or both PI and HPVG (4 patients). The rates of intestinal ischemia and mortality are given in Table 2. All 12 patients with positive radiographic findings underwent exploratory laparotomy: 10 (83%) patients had intraoperative evidence of bowel ischemia and 2 patients did not. One of these two patients had a repeat scan postoperatively due to a deteriorating clinical condition. The repeat scan showed progression of PI and significant HPVG; this patient later died from multiple organ failure. Postmortem examination revealed global bowel infarction and PI. In addition, one patient had a negative abdominal x-ray but later underwent a laparotomy because of rapid clinical deterioration. Extensive bowel ischemia was identified at laparotomy, and the patient died shortly thereafter. PI was diagnosed only on postmortem examination. This was the only false-negative radiographic study among the patients reviewed.

Only two patients in this study did not have radiologic studies performed before operative intervention. One of these patients arrested, developed a severely distended abdomen and had a positive diagnostic peritoneal lavage (gross stool), which lead to a laparotomy and identification of infarcted intestine. The other patient developed refractory hypoten-

Table 2. Radiologic findings in burned patients with PI and their relation to ischemic bowel

	Nonsurvivors	Survivors	IB Present (%)	Mortality Rate (%)
PI only (n = 6)	3	3	5 (83)	50
HPVG only (n = 2)	2	0	2 (100)	100
PI + HPVG (n = 4)	3	1	4 (100)	75
No findings (n = 1)	1	0	1 (100)	100
Total studies (n = 13)	9	4	12 (92)	69

IB, ischemic bowel; *HPVG*, hepatic portal venous gas; *PI*, pneumatosis intestinalis.

sion, rapidly uptrending lactate levels, and a tense abdomen that prompted surgical intervention because of concerns of an abdominal catastrophe. Extensive intestinal ischemia and infarction were noted at laparotomy.

Patients (94%) underwent exploratory laparotomy within a median time of 2.5 hours (interquartile range 2–3 hours) from identification of PI on radiographic studies or clinical suspicion of ischemic bowel. Thirteen patients were found to have ischemic or infarcted bowel at laparotomy. Thus, we observed a 13% negative laparotomy rate. Ten patients underwent bowel resection. Three others with ischemic bowel were deemed to be unresectable: two because of panischemia and infarction that would have required resection of the entire small and large intestine and one because of the extent of adhesions that made resection technically impossible. Two patients (13%) with positive CT scans underwent laparotomies that yielded findings of nonischemic, healthy appearing small bowel. All pathologic specimens (n = 14) showed evidence of PI either on gross or on histological inspection.

PI frequently involved both the small bowel and the colon (60%). Ischemia isolated to the small bowel alone was noted in four patients and the colon alone in only one patient. One of the surviving patients was found to have no gross intestinal ischemia at laparotomy. Of the four survivors, two patients had small bowel involvement only, one had both small and large bowel involved, and one patient had normal bowel at laparotomy. Ninety-one percent of nonsurvivors had infarction of long lengths (>30 cm) of small intestine or multiple large bowel segments, compared with none of the surviving patients (P = .004). Three of the survivors had pneumatosis and ischemia limited to short segments of intestine (Table 3).

Ninety-one percent of the nonsurvivors had open abdomens postoperatively compared with none of the survivors (P = .004). Table 4 illustrates perioperative factors that may have had effects on mortality. Nonsurvivors had greater base deficit (P = .03) and lactate levels in the 24-hour period preceding diagnosis. Lactates were available for 13 patients (10 nonsurvivors and 3 survivors). Nine nonsurvivors and one survivor had maximal lactate levels >2 mmol/L. In addition, 75% of the nonsurvivors had vasopressor treatment in the 24 hours before diagnosis of PI (P = .019). We found no statistical difference between nonsurvivors and survivors regarding time of initiation and rate of tube feeding. Enteral nutrition was initiated at a median of 3 days (2.5–4.5 days) postinjury in nonsurvivors and 3 days (2–5.25 days) in survivors. PI developed at a median of 6 (5–14) and 11 (9–13) days after initiation in nonsurvivors and survivors, respectively (P = .47). Nonsurvivors were receiving enteral nutrition at a median rate of 125 ml/hr (108–153 ml/hr) before the identification of PI, compared with survivors at a median rate of 88 ml/hr (73–104 ml/hr; P = .07). Ten patients (66%) were being fed with a 1.5 kcal/cc tube feed formula, whereas the remaining patients were fed with tube feed formulations ranging from 1.2 to 2.0 kcal/cc. Three of the 15 patients (20%) in this study were on both tube feeds and vasopressors at the same time (all were in the nonsurvivor group).

Table 3. Extent, location, and operative management of intestinal infarction based on operative, pathology, and autopsy data

	Nonsurvivors (n = 11)	Survivors (n = 4)	Overall (n = 15)	*P*
Extent of infarction				
No infarction	0	1	1	
Segmental infarction*	1	3	4	.03
Massive infarction†	10	0	10 (67%)	.004
Location of infarction				
Small bowel	10	3	13	.41
Large bowel	9	1	10	.07
Small and large bowel	8	1	9	.24
Operative intervention (n = 15)				
Bowel resection	7	3	10	
Unresectable‡	3	0	3	
No resection (no ischemia)	1	1	2	

* Infarction limited to 30 cm or less in the small bowel or limited to single segment of large bowel (ie, right colon).

† Extensive infarction of the small bowel (ie, entire jejunum, ileum, or both) or infarction of two or more large bowel segments (ie, right + transverse colon).

‡ Unresectable because of either treacherous intraabdominal adhesions or global ischemia incompatible with life.

DISCUSSION

The principal finding of this study was that PI was associated with intestinal ischemia in 93% of the patients in this study. Fifteen thermally injured patients (1.3%) admitted to our BICU over a 80-month period were retrospectively identified to have either ra-

Table 4. Perioperative data of patients with PI

	Nonsurvivors (n = 11)	Survivors (n = 4)	Overall (n = 15)	*P*
Vasopressor use*	9 (75%)	0	9 (56%)	.02
Base deficit at Dx	−5.4 (−13 to −4)	−1.7 (−0.1 to −3.1)	−4.4 (−9.5 to −2.3)	.03
Lactate level* (mmol/L)	4.1 (2.2 to 5.5)	1.7 (1.5 to 2.1)	2.6 (1.8 to 4.5)†	.05
Dx to surgery (hr)	2.5 (2 to 3)	2.5 (2 to 3)	2.5 (2 to 3)	.89
Surgical management	11	4	15 (100%)	
Negative laparotomy	1	1	2 (13%)	.48
Open abdomen‡	10	0	10 (67%)	.004
TF to PI (d)	6 (5 to 15)	11 (9 to 13)	7 (5 to 14)	.47
Rate of TF (ml/hr)	125 (108 to 153)	88 (73 to 104)	115 (95 to 148)	.07

Data are medians (25th and 75th interquartile range).

* Within 24 hr of PI development.

† Lactate levels missing from one survivor and one nonsurvivor (based on 13 patients).

‡ Nonapproximation of the fascia, either primarily (sutures) or secondarily (mesh).

TF, tube feeds; *Dx*, diagnosis; *PI*, pneumatosis intestinalis.

diographic or pathologic evidence of PI. This is the first case series of PI in the burn literature. Despite timely operative intervention, the overall survival rate was 27%. Those who survived were found to have limited intestinal infarction, and their abdomens were successfully closed postoperatively. In general, then, surgical intervention in patients with burn injury with PI is not futile, despite the risks associated with laparotomy after thermal injury.

Understanding the pathophysiology of PI is important before attributing it to an abdominal catastrophe. Three theories exist to explain the accumulation of gas in the intestinal wall. The "mechanical" theory postulates that the formation of submucosal gas may be caused by mucosal injury and loss of structural integrity, thus allowing intraluminal gas to dissect into the bowel wall.[12] Intestinal obstruction, severe ileus, blunt abdominal trauma, and endoscopy can increase intraluminal pressure forcing intestinal gas to enter the bowel wall.[12–14] The "microbiologic theory" suggests that the intraluminal bacteria are the origin of the intestinal wall gas.[15] Accordingly, intramural gas can accumulate in two ways: 1) pathogenic bacteria can enter the submucosa and begin producing gas if mucosal integrity fails[16] or 2) intraluminal bacteria produce hydrogen gas that diffuses through the mucosa and accumulates in the bowel wall.[17,18] The former is a more likely explanation for PI in patients with intestinal ischemia, because mucosal epithelial cells are highly susceptible to ischemia and could allow bacteria to escape the lumen and into the submucosa. The latter is a less likely reason for accumulation of intramural gas in cases of pathologic PI.[19] Finally, there are cases of PI attributed to air from ruptured alveoli, which tracts along mediastinal vascular structures, to the retroperitoneum, and then along the mesentery to accumulate in the subserosa.[15] In this study, almost all our cases were accompanied by ischemia, pointing to mechanical and microbiological factors. However, the causative factor of bowel ischemia in these patients remains uncertain.

The true incidence of PI is also unknown. A majority of patients from all populations with PI have benign intestinal pathology and remain asymptomatic. Typically, those patients with abdominal symptoms have PI identified as an incidental finding on radiologic evaluation.[12] Since the first description[1] of PI in the 18th century, the adult literature consists of sporadic case reports (>400) and a handful of review articles.[4,12,20] We found only six case series (including this one) discussing diagnosis, management, and outcomes.[5,21–24] PI has been identified in many patient populations, but there is a paucity of reported cases in the severely burned. To our knowledge, only two cases have been described: one adult in a retrospective study reviewing GI complications after thermal injury and one case report of a pediatric patients with burn injury.[8,10]

The management of PI remains controversial. The clinical significance is still unclear, and it is not pathognomonic of any specific disease process and can be seen with traumatic injuries, autoimmune disorders, inflammatory or infectious intestinal disease states, chronic pulmonary conditions, medications, and intestinal ischemia.[5,25] Many surgeons believe that PI equals intestinal infarction and requires urgent laparotomy, despite the identification of other intestinal diseases with similar radiographic findings.[14,21,22] They advocate urgent abdominal exploration because intestinal infarction carries with it an inherently high mortality rate, and abdominal exploration is the only way to rule it out.[26]

In our study, we found that patients with PI or HPVG identified on radiologic studies most often had underlying intestinal ischemia or infarction as the underlying causative factor. These findings prompted surgical intervention, and this was the primary management in 15 patients (100%) during their ICU admission. Of those patients undergoing abdominal exploration, 12 patients (80%) had PI or HPVG on radiographic studies and three other patients had clinical signs suggestive of intestinal ischemia. Intraoperatively, 13 were found to have intestinal ischemia or infarction with an operative mortality rate of 73%. This high mortality rate is similar to that found by Desai et al[7] and Markell et al[9] in thermally injured patients with intestinal ischemic complications.

Intestinal ischemia is difficult to diagnose in critically ill patients because physical examination findings may be unreliable; however, abnormal physical findings such as worsening abdominal distension, peritoneal signs, or hematochezia should prompt the physician to use radiologic evaluations in an attempt to identify it. We considered studies as "positive" if there was radiographic evidence of PI or HPVG. These findings were identified in 92% (n = 12) of these studies prompting urgent surgical exploration. Laparotomy revealed intestinal infarction in 10 patients (83%), and 2 patients in this group had no gross evidence of intestinal infarction. Our negative laparotomy rate was similar to those previously reported.[23,24] We found that radiologic evidence of PI or HPVG was often associated with intestinal ischemia or infarction in our patients. There are six case series in the literature discussing a correlation among radiologic findings, intestinal ischemia, and mortality in these patients.[5,21–24] The majority of these case studies had lower overall mortality rates and fewer patients with intestinal infarction compared with our study. Rather, the relationship we found between radiologic findings, intestinal infarction, and mortality corroborates the findings of Wiesner et al.[22]

Conversely, PI may be associated with negative findings at laparotomy. This was the case in two of our patients. One of these had benign disease (distended but healthy intestine) and recovered uneventfully. The other had no overt evidence of intestinal ischemia at surgery but died days later, only to have extensive infarction identified at autopsy, thus constituting a "false-negative" laparotomy. The latter case indicates that patients with a negative laparotomy may benefit from intraoperative intestinal imaging or a low threshold for a second-look operation.

Patients who develop intestinal ischemia often have a metabolic acidosis with increased serum lactate levels.[26] We were not surprised to find that nonsurvivors had higher lactate levels than did survivors (median 4.1 vs 1.7 mmol/L) and greater base deficits (median −7.1 vs −1.7) within the 24 hours before identification of PI. All 15 patients were admitted with base deficits that improved during their admission, only to worsen with the development of PI. We believe that PI in the presence of a severe metabolic acidosis, with increased lactate levels and worsened base deficits, may be due to intestinal ischemia. Our hypothesis is supported by Knechtle et al[5] who demonstrated that patients with increased lactate levels often had necrotic bowel and decreased survival compared with those patients with normal (<2 mmol/L) lactate levels. Furthermore, our hypothesis is supported by the findings of Hawn et al[21] who demonstrated that increased serum lactate levels (>2 mmol/L) is a positive predictor of ischemic bowel and increased mortality (positive predictive value 84%) in patients with radiographic PI. Therefore, we suggest that there should be a lower threshold for abdominal exploration in thermally injured patients with both CT scan evidence of PI and increased lactate levels.

PI can occur anywhere along the GI tract either in a primary (idiopathic) or in a secondary form. Eighty-five percent of cases are because of secondary PI. The literature describes the colon (50%) followed by the small bowel as the most common locations for PI.[5,21,23,27] This fact seems to only pertain to patients who develop primary pneumatosis, whereas the small bowel is the most common site in patients who have secondary PI.[4,20,28,29] In our series, we found that PI occurred most frequently in the small and large bowel concurrently (60%) compared with only 27% isolated to the small bowel and 7% only in the large bowel. This finding is contrary to the literature, and 100% of the patients in this study developed secondary PI because of intestinal infarction. This is likely to be the reason why small bowel was affected more often than large bowel in this patient cohort. All the patients in this study were found to have secondary PI. Another interesting finding in this study was a 90% mortality rate in patients found to have PI involving both the small and large intestine. It seems in this study that extensive pneumatosis because of pan-intestinal infarction is associated with increased mortality. This is supported by Hawn et al[21] who found similar outcomes related to the location of pneumatosis on CT scans. Conversely, Morris et al[23] had contradictory findings and believed that the location of pneumatosis alone has no prediction on overall outcome because of its rarity and the lack of large series, and it is hard to determine whether a relationship exists between the location of PI along the GI tract and subsequent mortality.

It is a common misconception that PI itself is associated with increased mortality rates; however, the

fact is that the underlying pathology causing the formation of pneumatosis is often the cause of mortality. We found in this study that there are several factors that could be associated with increased mortality in patients with burn injury with PI. First, we observed that vasopressor use was associated with increased mortality ($P = .02$). Eighty-two percent of the nonsurviving patients required vasopressors within 24 hours of developing PI. However, we cannot determine whether vasopressor use was the inciting agent that precipitated the development of intestinal ischemia or was required as a result of septic shock from intestinal infarction. There are multiple studies in the literature demonstrating that the use of vasoactive agents can alter both blood flow to and within the splanchnic vascular bed causing significant intestinal ischemia, infarction, and mortality.[19,26,30,31] In comparison, septic shock itself may lead to diminished blood flow to the splanchnic circulation, precipitate intestinal mucosal hypoperfusion, and possibly lead to intestinal infarction.[32] It was interesting that none of the surviving patients required vasopressor support before diagnosis of PI. We believe that vasopressors may be associated with an increase in mortality in patients with burn injury with ischemic bowel and that intestinal ischemia should be considered in the differential diagnosis of patients with burn injury with septic shock.

Second, we observed that the extent of infarction and even its location may be associated with increased mortality in patients with burn injury. Those thermally injured patients in this study with massive intestinal infarction had a 100% mortality rate, compared with a 25% mortality rate in patients with segmental infarction. Our findings corroborate those of Wiesner et al[22] that bowel infarction involving two or more bowel segments portends a mortality rate ranging from 71 to 100%. This is further validated by Goudet et al[33] who showed by multivariate analysis that the extent of infarction was significantly associated with increased mortality rates. Approximately 63% of the patients who underwent laparotomy for PI returned from the operating room with an open abdomen. It may be difficult to achieve primary closure in thermally injured patients because of the presence of bowel edema and constricting abdominal burns. Our observation of high mortality associated with the presence of an open abdomen in patients with burn injury was similar to that reported by Markell et al[9] and Hershberger et al,[34] who demonstrated that patients with open abdomens and burns >40% TBSA have dismal survival rates, even if they do not have ischemic bowel.

The main limitations of our study are the retrospective design, the use of a single burn center's data, and the small number of patients. The size of this patient cohort (n = 15) likely introduced type II error and other statistical limitations into the study. For example, known independent predictors of mortality, such as burn size, age, and inhalation injury, were found to be nonsignificant.

In conclusion, PI is an uncommon radiographic finding that is nonspecific and represents a broad spectrum of intestinal disease processes. The incidence in the general population is unknown and often been reported as individual cases, making its association with intestinal disease difficult. Burn patients can develop PI, but this has not been well documented in the literature. At our institution, we found that 1.3% of patients with thermal injuries admitted to the ICU had either radiographic or pathologic evidence of PI. Ninety-four percent of these patients were found to have ischemic bowel, and this translated to an overall mortality rate of 73%. We conclude that PI in patients with burn injury is highly suggestive of intestinal ischemia and possibly of progression to infarction. In addition, patients with increased lactate levels and base deficits, and vasopressor requirements in the 24-hour period before diagnosis, had worse survival rates.

Although there do not seem to be any optimal radiographic studies or serological markers for intestinal ischemia, this study suggests that thermally injured patients who develop tube feed intolerance, worsening abdominal distension, and increasing lactate levels should undergo radiologic evaluation to rule out intestinal ischemia (represented in this study by PI or HPVG). Furthermore, all survivors had relatively small ischemic areas that were successfully resected and underwent successful abdominal closure. Given these results, we recommend that patients with severe burn injury with radiographic evidence of PI or HPVG be considered immediately for abdominal exploration to exclude ischemic bowel.

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